

American College of Sports Medicine Roundtable on Exertional Heat Stroke — Return to Duty/Return to Play: Conference Proceedings

Francis G. O'Connor¹, Douglas J. Casa², Michael F. Bergeron³, Robert Carter III⁴, Patricia Deuster¹, Yuval Heled⁵, John Kark⁶, Lisa Leon⁴, Brendon McDermott⁷, Karen O'Brien⁸, William O. Roberts⁹, and Michael Sawka⁴

¹Military and Emergency Medicine, Consortium for Health and Military Performance, Uniformed Services University, Bethesda, MD; ²Korey Stringer Institute, Neag School of Education, University of Connecticut, Storrs, CT; ³Sanford School of Medicine of the University of South Dakota, Sioux Falls, SD; ⁴U.S. Army Research Institute of Environmental Medicine Thermal and Mountain Medicine Division, Natick, MA; ⁵Heller Institute of Medical Research, Sheba Medical Center, Tel-Hashomer, Israel; ⁶Department of Hematology, Thomas Jefferson University, Philadelphia, PA; ⁷Department of Health and Human Performance, University of Tennessee at Chattanooga, Chattanooga, TN; ⁸Command Surgeon, U.S. Army Training and Doctrine Command; ⁹Department of Family Medicine and Community Health, University of Minnesota, Minneapolis, MN

O'CONNOR, F.G., D.J. CASA, M.F. BERGERON, R. CARTER, P. DEUSTER, Y. HELED, J. KARK, L. LEON, B. MCDERMOTT, K. O'BRIEN, W.O. ROBERTS, and M. SAWKA. American college of sports medicine roundtable on exertional heat stroke — return to duty/return to play: conference proceedings. *Curr. Sports Med. Rep.*, Vol. 9, No. 5, pp. 314–321, 2010. On October 22–23, 2008, an ACSM Roundtable was convened at the Uniformed Services University (Bethesda, MD) to discuss return-to-play or return-to-duty for people who have experienced exertional heat illness (EHI) and to develop consensus-based recommendations. The conference assembled experts from the civilian sports medicine community and the Department of Defense to discuss relevant EHI issues, such as potential long-term consequences, the concept of thermotolerance, and the role of thermal tolerance testing in return-to-play decisions. Although the group was unable to move forward with new consensus recommendations, they clearly documented critical clinical concerns and scientific questions, including the following: 1) no uniform core definitions of EHI; 2) limited validated criteria to assess recovery from exertional heat stroke (EHS); and 3) inadequate ability to predict who may be predisposed to a subsequent heat injury after EHS. Areas of potential future research are identified.

INTRODUCTION

Exertional heat illness (EHI), specifically exertional heat stroke (EHS), continues to be a significant problem confronting athletes, coaches, and medical providers (12,47). In addition, EHI is a major concern for the military, particularly during recruit training, and remains a common cause of preventable nontraumatic exertion-related death (11,41,42). Some individuals with EHS experience long-term complica-

tions that may include multisystem organ (liver, kidney, muscle) and neurologic damage, as well as reduced exercise capacity and heat intolerance (12,52,57,69). Animal and human research suggest late or untreated EHS may result in organ damage that continues for weeks to months and possibly even after clinical symptoms or biomarkers have returned to normal (4,30,64). Whether this is true for rapidly treated EHS is not known. In the military, studies have demonstrated that an initial EHI episode during basic training does not predict or imply hospitalization for another EHI during subsequent military service. Importantly, occurrence of EHI during basic training only has a small impact on subsequent military retention and hospitalization (44). Although a recent epidemiological study of military cases suggested EHI may increase long-term mortality from organ failure (kidney, heart, liver), the timing and types of treatment for EHS were not considered (64). This evidence emphasizes the need to

Address for correspondence: Francis G. O'Connor, M.D., M.P.H., FACSM, Uniformed Services University, 4301 Jones Bridge Road, Bethesda, MD 20814 (E-mail: foconnor@usuhhs.mil).

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better characterize treatment protocols in all future studies of EHS episodes to accurately determine recovery and the effect of subsequent exercise-heat stress on vulnerable tissues. In addition, future studies must address long-term risks from other concomitant EHI insults, such as ischemia, oxidative, or nitrosative stress.

Because of the potentially damaging effects of EHS, one lingering question relates to return-to-play/duty. Current civilian and military return-to-play/duty guidelines largely are based on “best guess” estimates and clinical anecdotes, rather than valid biomarkers of recovery and definitive scientific evidence (1,12,43). Most organizations recommend return-to-play/duty after resolution of any abnormal clinical symptoms and a gradual increase in physical activity and exposure to heat stress (35). Although current American College of Sports Medicine (ACSM) recommendations suggest EHS casualties may return to practice and competition when the individual has demonstrated “heat tolerance,” defining heat tolerance and intolerance is an area of scientific controversy. The Israeli Defense Force (IDF) has employed a heat tolerance test (HTT) to evaluate fitness for return-to-duty for more than 30 yr; however, this approach was controversial among Roundtable panel members. The inability to accurately determine complete recovery after EHI/EHS negatively impacts both athletes and military force readiness. This article reports and discusses the conference proceedings from an ACSM Roundtable held to discuss the topic of EHI/EHS: return-to-play and return-to-duty. The specific purpose of this Roundtable was to outline and discuss the relevant issues, potential research, and current consensus recommendations.

METHODS/APPROACH

On October 22–23, 2008, an ACSM Roundtable was convened at the Uniformed Services University (USU, Bethesda, MD) to discuss the issue of when to return individuals (athletes and war fighters) who have experienced EHI to either play or duty and to develop consensus-based recommendations (Table 1). The conference assembled experts from both the civilian sports medicine community and the Department of Defense and in collaboration with the American Medical Society of Sports Medicine (AMSSM) and the National Athletic Trainers Association (NATA). It was structured into

TABLE 1. Roundtable educational objectives.

1. Review the definition, epidemiology, and pathophysiology of heat stroke
2. Review the diagnosis and management of exertional heat stroke
3. Describe current civilian and military guidelines that discuss return-to-duty/play as pertaining to heat stroke
4. Discuss the roles of thermal tolerance testing and genetic and biomarker evaluation in return-to-duty/play as pertaining to heat stroke
5. Describe the process of rehabilitation, clearance, and prevention in return-to-duty/play as pertaining to heat stroke
6. Construct a group consensus document that identifies current concepts with regards to return-to-duty/play as pertaining to heat stroke and outlines required areas for further research

seven 1-h topic blocks: definition and basic epidemiology, pathophysiology, recognition and treatment, thermal tolerance testing in recovery and return-to-play/duty, genetic and biomarker testing in recovery and return-to-play/duty, the role of sickle cell trait (SCT) in EHI, prevention of an ensuing incident of EHS, and current civilian and military guidelines for return-to-play/duty. At the conference conclusion, all speakers, discussants, and invited experts participated in a joint session to discuss areas of consensus and controversy and identify a potential “way ahead.” The speakers subsequently were asked to prepare a summary statement for each topic area to identify: what we know, what we do not know, and finally, where to go from here.

FINDINGS AND CONCLUSIONS

Definition and Epidemiology of EHS

What we know

1. EHS is defined as a multisystem illness characterized by central nervous system (encephalopathy), organ (*e.g.*, liver, renal), and tissue (*e.g.*, gut, muscle) dysfunction or injury associated with high body temperatures, generally $>104^{\circ}\text{F}$ (40°C) at time of insult, from strenuous exercise and/or environmental heat exposure (1,7,69).
2. The U.S. Army observed a fivefold increase in EHS hospitalization rates from $1.8 \cdot 100,000^{-1}$ in 1980 to $14.5 \cdot 100,000^{-1}$ in 2001. Gender, race/ethnicity, and geographic home of origin can explain much of the differences in EHS hospitalization rates in military populations (11,62), but approximately 50% of military EHS cases in basic training do not share these demographics and should, therefore, be considered moderate- to low-risk individuals (21).
3. Soldiers hospitalized for EHS subsequently had increased mortality (30 yr later) from cardiovascular, liver, renal, and gastrointestinal diseases compared with soldiers hospitalized for appendectomies (64). However, we do not know if, when, or how the individuals with EHS were treated when EHS was recognized.

What we do not know

1. Do differences in EHS definition change reported incidence rates?
2. What underlying factors account for the observed increases in EHS hospitalization rates among military and possibly civilian populations?
3. Do environmental factors, such as infection, and fatigue, compromise molecular protection and increase risk?
4. Does the use of selected categories of nutritional supplements — stimulants and thermogenics — contribute to increased incidence of EHS?

Where we go from here

1. Conduct a survey in sports medicine and athletic training communities regarding the knowledge of EHI to include “How do you define EHS?”

2. Conduct epidemiological studies to determine the intrinsic and extrinsic factors contributing to recent increases in EHS in athlete and military populations.
3. Monitor prospectively long-term organ damage in EHS patients based on the timing and type of cooling intervention.

Pathophysiology

What we know

1. Heat stroke often is classified as “classic” or “exertional,” with the former primarily observed in sick and compromised populations during heat waves and the latter primarily observed in apparently healthy and physically fit populations when engaged in physical exertion in varying environments (69).
2. Untreated EHS, compared with “classic” heatstroke, induces greater morbidity and mortality and more likely is associated with rhabdomyolysis, renal failure, liver damage, hyperkalemia, hypercalcemia, and hypoglycemia (69).
3. Environmental and exercise heat stress produce cardiovascular challenges as blood flow is diverted from viscera to skin and vital organs. Reduced perfusion of the intestine can result in ischemia and endotoxemia; this exposes organs to oxidative-nitrosative stress, hypoperfusion, ischemia, and hyperthermia. A systemic inflammatory response syndrome (SIRS) can be initiated and lead to multiorgan dysfunction (7,69).
4. Current theories regarding EHS include the “multiple hit” hypothesis, which identifies rapid hyperthermia (possibly mediated *via* the vagus nerve through endotoxin release and other factors) induced by compromised molecular protective mechanism in tissues and possibly mediated by prior viral exposure and other factors (60,61). In addition, endotoxin release is thought to lead to production of pyrogenic cytokines, which may compromise further normal thermoregulation (7).
5. Histological evidence of profound damage to liver, kidneys, intestine, spleen, and brain has been reported consistently in those who have not been treated in a timely manner. Brain injury appears to be most concentrated in the cerebellum, with evidence of Purkinje layer involvement (34). Other hyperthermia-induced brain dysfunctions likely include blood brain barrier breakdown, blood-cerebral spinal fluid barrier breakdown, serum protein leakage, and exacerbated drug-induced toxicity (27,34,58).

What we do not know

1. Do infection, muscle injury and/or other unknown factors prime the acute phase response and augment the hyperthermia of exercise?
2. Do any of these factors uncouple molecular protective mechanisms to induce unexpected EHS?
3. What are the molecular mechanism(s) of acquired thermal tolerance (ATT) and how might they be manipulated to improve tissue protection?

4. Does prior infection produce proinflammatory cytokines that deactivate the cells’ ability to protect against heat shock by negating ATT?
5. How long does the organ pathology and vulnerability persist after EHS, with and without immediate, rapid cooling?
6. What molecular biomarkers can be used to indicate full recovery after an episode of EHI/EHS?
7. What pharmacological interventions can be used to reduce morbidity and mortality from EHI/EHS and facilitate reset of ATT?

Where we go from here

1. Develop *in vivo* animal models of EHS (“classic” and “exertional”) to evaluate pharmacological interventions and return-to-activity guidelines.
2. Develop and evaluate pharmacological treatments in humans for EHS to reduce morbidity and mortality and accelerate return-to-activity.
3. Develop novel approaches to induce ATT and quantify molecular biomarkers of ATT induction and deactivation.

Recognition and Treatment

What we know

1. Promptly recognized and rapidly cooled EHS patients demonstrate both markedly improved survival and reduced complications (2,6,10,16,46,48). Unrecognized, untreated EHS often is fatal (46). Early season heat-related high-school football deaths occur most often in the first 1–4 d of practice, which emphasizes a need for acclimatization and on-site medical care to prevent fatal EHS (47).
2. The risk of EHI in susceptible individuals increases as wet bulb globe temperature (WBGT) rises (17,45,50).
3. Although EHS most commonly occurs in hot environments, it also can occur in relatively cool conditions (<10°C WBGT) (48,49).
4. Protective equipment worn by football players and military personnel reduces the ability to dissipate heat from the body and lowers the tolerable WBGT level that is safe for full activity (28).
5. Rectal temperature is the only consistently reliable and valid way to measure core temperature in the field (3,9,15,17,53). However, cooling never should be delayed in an individual suspected of EHS where rectal temperature monitoring is not possible.
6. Whole-body, cold-water immersion with the water aggressively stirred will induce the fastest cooling rates for treating EHI/EHS (6). Whole-body cold-water accompanied by ice massage of major muscle groups also is successful, although cooling rates are not as high as with cold-water immersion (35).

What we do not know

1. What are the earliest signs and symptoms that are harbingers of impending EHS?

2. How long is the “golden hour” in EHS recognition and treatment?
3. What is the optimum body cooling end point for treating EHS?
4. How are cooling rates of EHS patients influenced by the specific anthropometric characteristics of the individual?

Where we go from here

1. Maintain treatment logs with temperature measures and resolution of central nervous system dysfunction during EHS treatment; this will allow for better understanding of the time sequence and prognosis in those who survive and those who do not.
2. Investigate the utility of cooling to lower temperatures than currently recommended (38.3°–38.9°C), *i.e.*, therapeutic mild hypothermia as a treatment end point.

Physiological Recovery

What we know

1. Severity of and presumably recovery from EHS depends upon proper, acute treatment for each body organ system or tissue affected.
2. Morbidity and mortality are a direct result of ischemia, oxidative, and nitrosative stress. The prognosis is worse in cases when T_{core} remains above the critical threshold of 40.5°–41.0°C (105°–106°F) for any period of time and early intervention is delayed (13,23,38).
3. Cardiovascular function, if impaired, usually recovers within hours following EHS (15,26,54–56). However, depending on the treatment, EHI may increase long-term mortality from organ failure, including the heart (64).
4. Hepatic tissue biomarkers may show hepatic stress for 24–48 h prior to returning to normal values (4,46,59). In the absence of appropriate treatment and cooling, hepatic tissue injury may persist for weeks to months after traditional biomarkers have returned to normal (4). Whether this is true for rapidly treated EHS is not known. Also, renal failure is common with EHS and may require weeks for recovery (7). As previously described, EHI may increase long-term morbidity from organ failure, to include the liver and kidney, in the absence of adequate rapid cooling (64).
5. Systemic biomarkers of musculoskeletal damage with associated rhabdomyolysis (creatinine kinase [CK], myoglobin) may increase for 24–96 h prior to returning to normal values (25,33,36).
6. Central nervous system function generally recovers rapidly in most survivors who are immediately cooled; however, additional evidence suggests severe EHS can result in significant, and often permanent, neurologic complications (33,36,52).

What we do not know

1. Can systemic biologic markers or a HTT identify recovery or future risk following EHS?

2. Altering what (if any) inherent mechanisms (and how) can facilitate recovery beyond immediate treatment of the patient?

Where we go from here

1. Research biologic markers and/or testing protocols of recovery in a wide range of EHI and EHS patients with the goal being able to identify primary indicator(s) to assist in clinical decision-making following EHS.
2. Evaluate nutritional, medical, and physiological strategies to facilitate recovery and potentially shorten the amount of time lost in military training and/or athletic competition.
3. Determine whether biologic markers of systemic damage indicate functional deficits that hinder return-to-activity and the significance of delayed markers in evaluating immediate damage.

Thermal Tolerance Testing

What we know

1. The IDF currently uses a HTT for assessing heat intolerance post EHS and guiding return-to-duty decisions (39,40).
2. The IDF HTT is based upon the assertion that tolerance to heat stress varies among individuals. Individuals who are unable to tolerate a specific heat challenge, as indicated by someone whose body temperature rises earlier and at a higher rate than others, under the same environmental and exercise conditions, are defined as “heat intolerant” (39).
3. Many factors underlying heat intolerance are acquired and can permanently or temporarily affect the thermoregulatory response as demonstrated during the HTT (18).
4. Some evidence indicates EHS patients may be at higher risk for another EHS event (19,24,51,59).
5. Although the IDF has safely used the HTT to return soldiers to duty for over 30 yr, some in the Roundtable panel questioned the validity of this test for return-to-duty clearance.

What we do not know

1. Does the level of heat intolerance, as defined by the HTT, predict who will experience a subsequent EHI/EHS?
2. Does EHS induce an altered ability to thermoregulate, and if so, for how long?
3. How can deficits within the thermoregulatory system be quantified?
4. Is heat intolerance following EHS and demonstrated by HTT a reflection of a prior EHS or preexisting susceptibility?
5. Can the results of an HTT in a recent EHS patient dictate/predict appropriate physical activity progressions during the rehabilitation process?

Where we go from here

1. Determine mechanisms altering thermoregulation, as measured by HTT after EHS.

2. Improve understanding of the etiology of heat intolerance.
3. Develop a valid, safe, and simple field test to predict heat tolerance and potentially a risk profile for susceptibility to another EHS.

Genetic Testing and Biomarkers

What we know

1. Thermoregulatory responses during EHS recovery consist of hypothermia and/or recurrent fever in both human and animal models (32,34). Although these T_{core} responses are thought to reflect brain injury, or influence the hypothalamus, which may lead to thermoregulatory “instability,” actual damage to the hypothalamus has been undetectable in 125 fatal cases of heat stroke (34).
2. Thermoregulatory profiles of heat-stroked mice during recovery indicate hypothermia is a regulated response to protect against tissue injury (32,68).
3. Cytokines are likely key mediators of heat-induced SIRS (7,29). High circulating levels of pro- and antiinflammatory cytokines correlate with morbidity/mortality in EHS patients and animal models of heat stroke (5,7,8,14,22,31).
4. Cytokine knockout mice show enhanced heat stroke mortality (30).
5. High interferon-inducible gene expression suggests that prodromal (viral) illness may predispose to EHS susceptibility (61).

What we do not know

1. How accurately do gene and protein expression profiles measured in the circulation reflect injury at the tissue level?
2. Are circulating proteins (e.g., cytokines) appropriate biomarkers of tissue injury and recovery of tissue function?
3. Do monokines released by skeletal muscle during exercise serve a role in EHS?
4. What is the time course of tissue injury and cellular reset/recovery after EHS?
5. Does restoration of organ function to a homeostatic level reflect the ability of that organ to respond to a subsequent heat-related event?

Where we go from here

1. Animal models are needed to study the time course of thermoregulatory, cardiovascular, liver, kidney, and other tissue injury changes during recovery from EHS.
2. The contribution of skeletal muscle to the cytokine milieu needs to be further delineated with respect to EHS.
3. The efficacy of therapeutic treatments for EHS recovery needs to be evaluated in conscious animal models.
4. The relationship between circulating biomarkers and changes in specific cell populations at the tissue level (e.g., Kupffer cells, hepatocytes, splenocytes) needs to be delineated during EHS recovery to advance targeted therapeutics.
5. The protective function of heat-induced hyperthermia and/or fever requires elucidation.

6. The potential of common over-the-counter medications, such as nonsteroidal antiinflammatory drugs (NSAID), to compromise hypothalamic and liver function, exacerbate tissue injury, and/or impede tissue recovery must be studied.

Sickle Cell Trait

What we know

1. Sickle cell trait (SCT) is characterized by the inheritance of a normal hemoglobin gene (HbA) from one parent and an abnormal, mutated B1-globin sickle hemoglobin gene (HbS) from the other parent.
2. The prevalence of SCT in the United States is estimated at approximately 8% in African-American people and 0.05% in Caucasian people (63).
3. The literature has documented numerous cases of exertional sudden death in African-American soldiers in basic training and athletes with SCT. Contributing factors appear to include dehydration, exertion in the heat, high-intensity exercise, and exertion-related acidosis, any of which could lead to polymerization of HbS, as well as immune and vascular responses, to result in subsequent vascular occlusion and related consequences (63).
4. Fewer exercise-related deaths have been reported for soldiers participating in advanced specialty training after graduation than from recruit training; this suggests those who are susceptible have either been eliminated from the at-risk pool or have been placed in less susceptible settings (20,21,44,66).

What we do not know

1. What portion of EHS-related deaths in military recruits is independently a result of SCT?
2. Does the risk of serious complications from EHI among recruits with SCT positively correlate with the degree of hyposthenuria?
3. Are SCT or EHS deaths inadvertently attributed to unrecognized cardiac abnormalities or vice versa?
4. Does SCT predispose to EHI or EHS?

Where we go from here

1. Examine the relationship of exercise duration and intensity under unique environmental conditions to develop field profile responses for the SCT and normal hemoglobin populations. This will assist in understanding the relationship of exercise intensity and duration to potential physiological responses and risk.
2. Utilize risk profiles from prior heat exposures to modify exercise intensity and duration for military personnel, regardless of hemoglobin type.
3. Determine the effectiveness of reducing the daily exercise load based on the prior day's temperature (24°C or some other temperature) or a combination of consecutive day heat exposures.
4. Investigate the role of other single-nucleotide polymorphisms (SNP) in exercise-related sudden death and SCT in recruit populations to test whether other gene

variants are associated with risk of death in those who have experienced life-threatening EHI. Particular attention should focus on genes associated with anesthesia-related malignant hyperthermia and/or other genes related to potential mechanisms of EHI.

5. Perform more accurate population studies to define the risk of exercise-related sudden cardiac arrest events and deaths with and without SCT for military populations at different stages of their career.
6. Investigate the association between urine-specific gravity and SCT as a function of EHI.

Prevention of EHS

What we know

1. Individual risk factors for EHS include age >40 yr, medications (anticholinergics, antihistamines, stimulants to include medication for attention deficit/hyperactivity disorder, angiotensin-converting enzyme [ACE] inhibitors, and diuretics), skin disease (eczema, poison ivy, skin graft, burns), acute illness, chronic medical conditions that cause autonomic dysfunction, dehydration, poor acclimatization, high body mass index, use of selected dietary supplements, and/or poor conditioning (21).
2. Heat strain in response to heat stress is cumulative (65).
3. Coaches, trainers, and leaders can mitigate EHS risk by altering clothing, intensity, and duration of activity, rest periods, and/or environmental heat load (20).
4. Adherence to work-rest cycles, acclimatization protocols, and hydration guidelines improves heat tolerance (37,67).

What we do not know

1. What prevention mitigation measures are most effective for EHS?
2. What are the best techniques to “microclimate” cool athletes and soldiers *e.g.*, undergarment cooling vests?
3. What is the role of autonomic instability in EHS?
4. Does reestablishing autonomic stability improve EHS morbidity and mortality?

Where we go from here

1. Develop individual cooling devices that can sustain training and allow rapid rehabilitation when mild symptoms of heat stress are noted.
2. Compare and contrast the encephalopathy of EHS and concussive injury.
3. Determine whether medications that improve autonomic instability in the acute management of EHS improve long-term outcomes.

Current Guidelines for Return-to-Play

What we know

1. No comprehensive and validated guidelines or recommendations exist for returning athletes/soldiers to play/duty (35,43).

2. Most guidelines are “common sense” recommendations that require return to an asymptomatic state, return to normal laboratory parameters, and a cautious reintroduction to physical activity to ensure acclimatization (35).
3. The current recommendations from the ACSM for returning an athlete to training and competition are (1):
 - a. “Refrain from exercise for at least 7 d following release from medical care;
 - b. Follow up about 1 wk postincident for physical examination, and lab testing or diagnostic imaging of affected organs based upon the clinical course of the heat stroke incident;
 - c. When cleared for return-to-activity, begin exercise in a cool environment and gradually increase the duration, intensity, and heat exposure over 2 wk to demonstrate heat tolerance and to initiate acclimatization;
 - d. If return to vigorous activity is not accomplished over 4 wk, consider a laboratory exercise-heat tolerance test;
 - e. Clear the athlete for full competition if heat tolerant after 2 to 4 wk of full training.”

What we do not know

1. How should we quantify the magnitude and persistence of organ injury from EHS?
2. What are the long-term health implications of EHS?
3. What clinical aids can a physician use to assess recovery from EHS?
4. How can we ascertain that an athlete/soldier has fully recovered from an EHI?

Where we go from here

1. Ascertain the time course of clinical and physiologic recovery from EHS.
2. Determine the risk for subsequent EHI after one EHI episode.
3. Develop regional centers to assist in making difficult clinical decisions on athletes/soldiers who have had EHS.
4. Develop and implement a strategy for clinical and physiologic research in the area of environmental illness to improve the current evidence base.
5. Develop better clinical tools, educational protocols, and practice guidelines for returning EHS patients to the field of play/duty.

CONCLUSION

The principal goal of the ACSM Roundtable discussion was to develop a consensus document reflecting the best guidance to return athletes and soldiers to activity following EHI, in particular, EHS. Although the conference succeeded in discussing relevant issues, such as the potential long-term consequences of EHI, and current controversies, such as the concept of thermotolerance and the role of thermal tolerance testing in assisting in return-to-play decisions, the assembled attendees were unable to move forward with new consensus recommendations. The conference proceedings, however, clearly documented critical clinical concerns

TABLE 2. Army medical department exertional heat illness definitions for the purpose of medical profiling.

Heat exhaustion (HE) is defined as a syndrome of hyperthermia (core temperature at time of event usually $\leq 40^{\circ}\text{C}$ or 104°F) with physical collapse or debilitation occurring during or immediately following exertion in the heat, with no more than minor central nervous system (CNS) dysfunction (e.g., headache, dizziness). HE resolves rapidly with minimal cooling intervention

Heat injury (HI) is defined as HE with clinical evidence of organ (e.g., liver, renal, stomach) and/or muscle (e.g., rhabdomyolysis) damage without sufficient neurological symptoms to be diagnosed as heat stroke

Heat stroke (HS) is defined as a syndrome of hyperthermia (core temperature at time of event usually $\geq 40^{\circ}\text{C}$ or 104°F), physical collapse or debilitation, and encephalopathy as evidenced by delirium, stupor, or coma, occurring during or immediately following exertion or significant heat exposure. HS can be complicated by organ and/or tissue damage, systemic inflammatory activation, and disseminated intravascular coagulation

and scientific questions that persist at this time. These questions limit the development of grade A, scientific, evidence-based guidelines for return-to-play/return-to-duty. Critical concerns of Roundtable members were the following:

- A lack of consensus on the core definitions of EHI
- Limited scientifically valid criteria to assess recovery from EHS
- Inadequate scientifically valid criteria to predict who may be predisposed to a subsequent heat injury after EHS

As a direct result of this Roundtable, military attendees in conjunction with civilian experts were able to hold further discussions relative to the dilemmas and issues previously identified. The U.S. Army medical department, in June of 2009, subsequently moved forward with new definitions (Table 2) and recommendations for soldiers who sustain an exertional heat injury (www.champ.usuhs.mil/chclinicaltools.html). These guidelines reflect the current best evidence and are based on “common sense” recommendations: they require “return to an asymptomatic state,” normal lab parameters for key end organ function, and cautious and graduated reintroduction to activity to ensure acclimatization. The currently available science cannot support a high-level, evidence-based, consensus return-to-play document, and much work has yet to be accomplished. The panel strongly believes this document provides a strong foundation for future clinical and bench research in this vitally important area.

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